September 10, 2007

Joshua Marx, Regulatory Project Manager
US Army Corps of Engineers
Kansas City District

Re: Permit No. 2006-1014

Applicant: BNSF Railway Company
4515 Kansas Avenue
Kansas City, KS 66106

Dear Mr. Marx,

I am a homeowner in close proximity to the proposed BNSF Railway facility. I have many concerns regarding the proposed facility and wish to state that I am adamantly against the proposed project. The City of Gardner and majority of its elected officials are in no way looking out for the best interests of the community, nor do they represent the opinions of the vast majority of citizens impacted. The most obvious solution to address the impacts addressed below would be to relocate the proposed facility a safe distance from populated areas. The following areas of concern are reasons why I believe this proposed facility should not be allowed:

Drinking water: Hillsdale Lake supplies a large population including many communities in the KC metropolitan area. The BNSF facility will greatly impact Bull Creek and many of its tributaries. Bull Creek is the primary tributary into Hillsdale Lake. Pollutant threats to the lake that currently exist include nitrogen, phosphorus, sediment and pesticide impairment. The proposed facility will subject Hillsdale Lake, our drinking water source with a multitude of organic contaminants and metals. Within EPA are numerous RCRA remediation and superfund projects documenting BNSF sites listing many of these contaminants. We have been told by BNSF and city representatives that new technologies will prevent this from happening. Regardless of the new technologies in place this facility still poses a great risk to the water resources that will impact thousands of area citizens. Monitoring and enforcement of facility operations, and water quality efforts will also subject the community to higher costs.

Stormwater Runoff: Runoff from the area covered by impervious surfaces will result in significant degradation and contamination of the primary tributary to Hillsdale Lake, Big Bull Creek. The stormwater runoff will carry solvents from maintenance operations, cleaning agents, fuels, additives and other contaminants. Stormwater basins are not a cure all system that will prevent these contaminants from impacting surface waters off site. Additional sediment and nutrient loads will also be created, increasing the current Clean Water Act impairment issues which the Hillsdale Lake watershed is attempting to address. Rerouting of the unnamed tributary will also increase sediment and nutrient loading, increasing the likelihood that the water quality standards will not be met for lake phosphorus levels.

Wetlands: Rail yard sites have become superfund sites, resulting from the use of contaminated fill materials brought in for use in site construction.

Groundwater: Residents adjacent to the facility utilize groundwater as their primary drinking water source through wells. A complete groundwater delineation study should be required to prevent the contamination of private drinking water. Monitoring wells must be required for early detection.

***Air quality impacts*:** Air quality impacts will be felt by the entire KC metropolitan area. Traffic Studies conducted by the DOT on the proposed BNSF Logistics Park provide adjusted growth forecasts, calling for significant infrastructure improvements including; the need for six lanes on I-35 north of Gardner Rd., US-56 will be operating above capacity, need for widening to 4 lanes, need for other significant improvements in the immediate area including a new I-35 interchange. Figures are based upon 59,809 trips per day, 4,003 truck trips per day. The figures provided by BNSF are much lower than should be expected.

Two significant impacts will be caused by the increased traffic and rail yard equipment, creation of ozone, resulting in non-attainment of the Clean Air Act for the Kansas City Metropolitan Area, and secondly increased concentrations of diesel particulate matter. Each of these impacts will result in adverse health impacts upon the area citizens as documented in numerous medical journals and research papers due to the close proximity of the community (Attachments). Of particular concern are the new public schools directly north of the proposed facility, with the prominent wind direction being from the south.

Safety and Security: Many hazardous materials are transported by the containers utilized by the proposed facility. Spills are common occurrences at rail yard sites. The types of hazardous materials transported by containers are also a concern of area citizens. Radioactive materials are shipped by containers; these types of materials must not be allowed in close proximity to a large populated area. Weapons of mass destruction are also a threat that should not be ignored. Terrorists are well aware of our shipping methods and could utilize this as a means to strike Middle America. For these reasons perimeter security is also a concern.

Park Impacts: The proposed facility is adjacent to a new county park in which Johnson county tax payers footed an enormous bill to benefit from new recreational opportunity. How many will now utilize this new park impacted by the aesthetics of a rail yard facility, noise of locomotives and rail yard equipment, and the runoff of pollutants into a re-routed stream that runs through the proposed facility and into the park. Not likely a good source of fish to take home and eat.

Economic Impact: Land and property values in the surrounding area have already been negatively affected. Only those owning land / property on and in the immediate area surrounding the facility have benefited from BNSF buyouts. The vast majority of area residents such as myself suffer the losses due to the greed of city, county and state officials waiting for increased tax revenues.

Sincerely,

Damon Frizzell
35335 W. 167th St.
Gardner, KS 66030

EXECUTIVE SUMMARY

For the "Proposed Identification of Diesel Exhaust as a Toxic Air Contaminant"

Prepared by the Staff of
the Air Resources Board and
the Office of Environmental Health Hazard Assessment

As Approved by the Scientific Review Panel
on April 22, 1998

Preface

This draft report has been released to solicit public comments on the revised "Public Comment and SRP Version" report which includes the Executive Summary, Part A (exposure assessment), Part B (health assessment), and Part C (responses to public comments) documents. This version of the report, along with the comments received and any revisions resulting from the comments, will be reviewed and discussed with the Scientific Review Panel (SRP) at a noticed public meeting of the Panel. It has been revised since its original June 1994 release and subsequent May 1997 release to incorporate public comments received during the first public comment period (June 1994-December 1994), second public comment period (May 1997-August 1997) and at the September 1994, January 1996, and July 1997 public workshops. It is important to note that we have updated this draft report to include the new EMFAC7G1.0 motor vehicle emissions inventory model results. This information is incorporated into our outdoor ambient exposure, indoor, and total exposure estimates. This information was presented at the July 1997 workshop.

The Air Resources Board's (ARB) consideration of diesel exhaust as a TAC will be made following this public comment period and review by the SRP. If the SRP approves the report, it will be presented to the ARB at a duly noticed public hearing, after a 45-day public comment period. If the ARB approves the report at a hearing and identifies diesel exhaust as a TAC, the information contained in the report will be used in the assessment of the need for additional control measures. While no new control measures specific to the toxicity of diesel exhaust are being proposed at this time, a number of existing sources of diesel engine exhaust are already subject to California regulations requiring reductions of criteria air pollutants contained in diesel exhaust. Any consideration of control measures to reduce exposures to diesel exhaust, if identified as a TAC, will be made only after a thorough public process including public workshops.

EXECUTIVE SUMMARY

For the "Proposed Identification of Diesel Exhaust as a Toxic Air Contaminant"

California Environmental Protection Agency
Air Resources Board
Office of Environmental Health Hazard Assessment

As Approved by the Scientific Review Panel on April 22, 1998

Introduction

The public's exposure to toxic air contaminants is a significant environmental health issue in California. In 1983, the California Legislature enacted a program to identify the health effects of toxic air contaminants (TAC) and to reduce exposure to these contaminants to protect the public health (Assembly Bill 1807: Health and Safety Code sections 39650-39674). The Legislature established a two-step process to address the potential public health effects from TACs. The first step involves determining if a substance is toxic and to what extent. This step is the risk assessment (or identification) phase. Under state law, the Air Resources Board (ARB) is authorized to identify a substance as a TAC if it determines the substance is "an air pollutant which may cause or contribute to an increase in mortality or an increase in serious illness, or which may pose a present or potential hazard to human health (Health and Safety Code section 39655)." An air contaminant means "any discharge, release, or other propagation into the atmosphere and includes, but is not limited to, smoke, charred paper, dust, soot, grime, carbon, fumes, odors, particulate matter, acids, or any combination thereof (Health and Safety Code section 39013).

The second step, determining the need for and appropriate degree of control measures, occurs only if the ARB identifies the substance as a toxic air contaminant. This step is the risk management (or control) phase of the process.

The ARB is evaluating diesel exhaust as a candidate toxic air contaminant under the State's air toxics identification program. This report presents the information upon which this assessment is based.

What is Contained in This Report?

This report consists of an Executive Summary which summarizes the scientific basis for the proposed identification of diesel exhaust as a TAC, and Parts A, B, and C of the Technical Support Document. Part A, prepared by the ARB staff, is an evaluation of emissions of, and exposure to, diesel exhaust. Part B, prepared by the Office of Environmental Health Hazard Assessment (OEHHA) staff, assesses the health effects. Part C consists of copies of the public comment letters and ARB/OEHHA's staff responses to public comments received on the May 1997 draft report.

This report does not address the need for, or contain any assessment of, or recommendations for, control measures to reduce exposure to diesel exhaust.

How Does the ARB Identify a Substance as a TAC?

With input from the public, industry, and the scientific community, the ARB and the OEHHA gather all of the relevant scientific information on a substance. Under the requirements of law (Health and Safety Code sections 39660-39662), the ARB and OEHHA must answer the following questions:

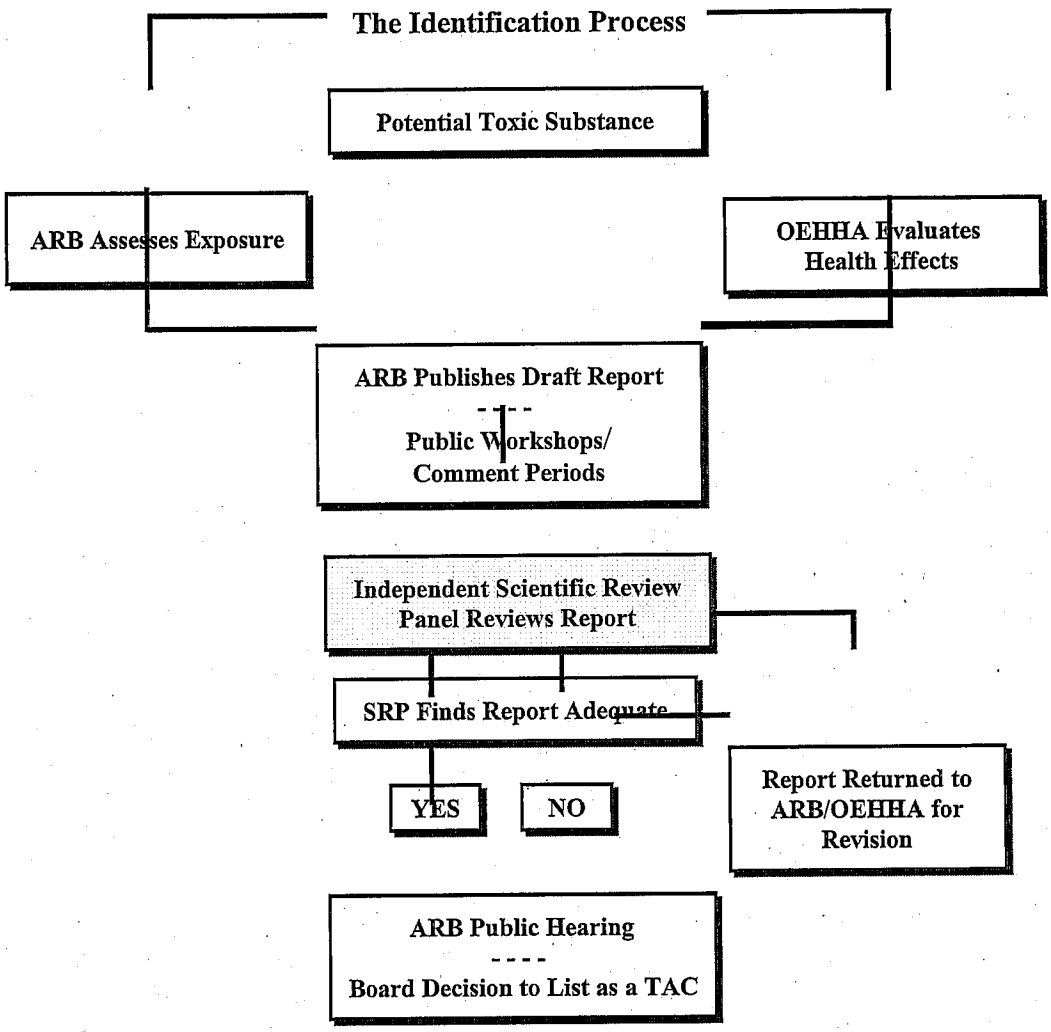
❶ Is the substance used in California?
❷ Who is exposed to the substance?
❸ How many people are exposed?
❹ How much is emitted into the air?
❺ How long does the substance stay in the air?
❻ How much of a substance can be measured in the air?
❼ Does the substance pose a potential health risk to Californians?

The ARB staff determines the public's potential exposure to the substance while the OEHHA must determine if the substance poses a potential health risk. Both agencies then prepare a draft report which serves as the basis for identifying a substance.

Once the draft report is released, the public review process begins. The public review is a critical step in identifying a substance. After the release of the report, a workshop is held to discuss the report during a formal comment period. After receiving public comments, both verbal and written, we carefully review all comments, incorporate new information, and revise the report where appropriate. Further workshops are held and revised reports are released for review, as needed.

After the comment period, the report is then submitted to the Scientific Review Panel (SRP). The SRP is an independent group of scientists, who review the report for its scientific accuracy. If the SRP determines that the report is not based on sound scientific information, it is sent back

to the staff for revisions. If the SRP approves the revised report, the SRP prepares its "findings" which are submitted, along with the staff report, to the ARB for consideration at a public hearing. The Board then decides whether to identify a substance as a toxic air contaminant (see illustration below). If the substance is identified as a TAC it is listed in Title 17 of the California Code of Regulations under section 93000.



What Happens When a Substance is Identified as a TAC by the Air Resources Board?

After a substance is identified as a TAC, the ARB conducts a needs analysis to determine if any regulatory action is necessary. Specifically, the law requires the ARB to prepare a report which assesses the need and appropriate degree of control of a TAC, in consultation with the local districts, affected industry, and the public. The report is required to address the following issues:

1. present and potential future emissions, and the associated risks,
2. physical and chemical characteristics,
3. number and categories of emission sources,
4. available control technologies,
5. associated costs for reducing emissions,
6. substitute compounds/pollution prevention,
7. potential adverse health, safety, or environmental impacts associated with the implementation of a control measure.

If reductions in exposure are needed, the ARB must design control measures that consider the above issues and reduce emissions to the lowest level achievable through the application of best available control technology or a more effective control method. Public outreach is an essential element in the development of a control plan and any control measures. In the course of this assessment, the ARB works with local districts and holds numerous public workshops and individual meetings with industry representatives in an open public process.

In the case of diesel exhaust, there have been several regulations enacted that have resulted or will result in significant reductions in emissions of pollutants such as NO_x, SO_x, and particulate matter from diesel engines (see pages ES-8 and ES-9). In the risk management phase, the adequacy of these current and future diesel exhaust control measures would be examined as part of the needs assessment.

Where is Diesel Exhaust in the Toxic Air Contaminant Process?

Diesel exhaust is in the development phase of the identification process. It has gone through an extensive evaluation since it entered into the identification program in October 1989. In March 1990, the ARB sponsored a conference on the risk assessment of diesel exhaust.
On June 17, 1994, the first draft report was released to the public for a six month comment period. On September 14, 1994, a public workshop was held to discuss the report. On January 29-30, 1996, the OEHHA, ARB, Health Effects Institute, National Institute for

Occupational Safety and Health, World Health Organization, and the United States Environmental Protection Agency (U.S. EPA) sponsored a scientific workshop to discuss the

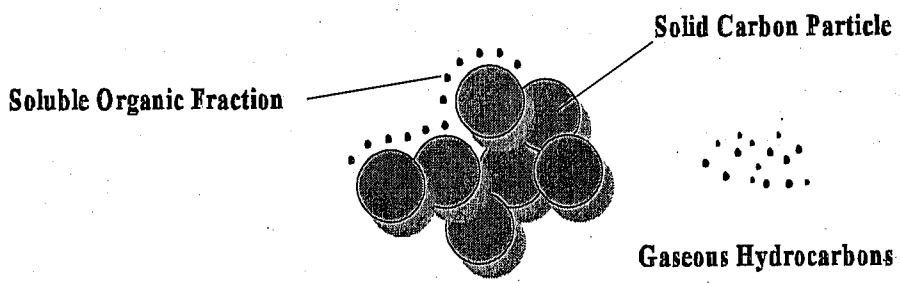
application of human health study data in developing quantitative cancer risk estimates for diesel exhaust. A second version of the draft report was released for public comment in May 1997. On July 1, 1997, a third public workshop was held to discuss the revised draft version of the report.

This version of the report has been released to the public for a third comment period. It reflects the public comments received on the exposure and health assessments during the first and second public comment periods and at the September 1994, January 1996, and July 1997 workshops. Currently, the SRP is planning on holding a special public meeting in March 1998 to hear from invited scientists who have expertise in the study of diesel exhaust to present their research and perspectives. It is the SRP's view that the material presented and discussed at this meeting will assist in a better understanding the science regarding the health effects of diesel exhaust. After this meeting and the end of the third comment period, the report, along with the comments received and any revisions resulting from the comments, will be formally discussed with the SRP at a duly noticed meeting. We anticipate that this meeting will occur in late April 1998. If the SRP approves the report, the report, and a proposal to formally list diesel exhaust as a TAC, will be presented to the ARB at a public hearing, after a 45-day comment period.

What is Diesel Exhaust?

Diesel exhaust is a complex mixture of thousands of gases and fine particles emitted by a diesel-fueled internal combustion engine (see figure below). The composition will vary depending on engine type, operating conditions, fuel composition, lubricating oil, and whether an emission control system is present.

The gaseous fraction is composed primarily of typical combustion gases such as nitrogen,



oxygen, carbon dioxide, and water vapor. In addition, the gaseous fraction also contains air pollutants such as carbon monoxide (CO), sulfur oxides (SO_x), nitrogen oxides (NO_x), volatile hydrocarbons, and low-molecular weight polycyclic aromatic hydrocarbons (PAH) and PAH-derivatives. Some of these gaseous components such as benzene, formaldehyde, 1,3-butadiene, arsenic, and nickel, are suspected or known to cause cancer in humans.

One of the main characteristics of diesel exhaust is the release of particles at a relative rate of about 20 times greater than from gasoline-fueled vehicles, on an equivalent fuel energy basis. The particles are mainly aggregates of spherical carbon particles coated with inorganic and organic substances. The inorganic fraction primarily consists of small solid carbon (or elemental carbon) particles ranging from 0.01 to 0.08 microns in diameter. The organic fraction consists

of soluble organic compounds (soluble organic fraction) such as aldehydes, alkanes and alkenes, and high-molecular weight PAH and PAH-derivatives. Many of these PAH and PAH-derivatives have been found to be potent mutagens and carcinogens (see Chapter III, section E). In a recent study, researchers identified a new class of potent mutagenic compounds in the organic extracts of both diesel exhaust and airborne particles. The results showed that the mutagenicity of this new class of compounds (nitrobenzanthrones), specifically, 3-nitrobenzanthrone, compared similarly with that of 1,8-dinitropyrene, which is one of the strongest direct acting mutagens previously discovered. Studies have shown that, depending on the condition of the engine and test cycle, the contribution of organics to the total diesel particulate matter mass could range from 10 to 90 percent.

Almost all of the diesel exhaust particle mass is in the fine particle range of 10 microns or less in diameter. Approximately 98 percent of the mass of these particles are less than 10 microns in diameter, 94 percent less than 2.5 microns in diameter, and 92 percent less than 1 micron in diameter. Consequently, because of their size, these particles can be inhaled and eventually trapped into the bronchial and alveolar regions of the lung.

Diesel exhaust includes over 40 substances that are listed by the U.S. EPA as hazardous air pollutants and by the ARB as toxic air contaminants. The substances listed below have either been detected or predicted to occur in the exhausts of diesel engines based on observed chemical reactions and/or their presence in the fuel or lubricating oil. Further research is needed to determine the contribution of many of these substances to atmospheric diesel exhaust exposures.

Substances in Diesel Exhaust Listed by the ARB as Toxic Air Contaminants

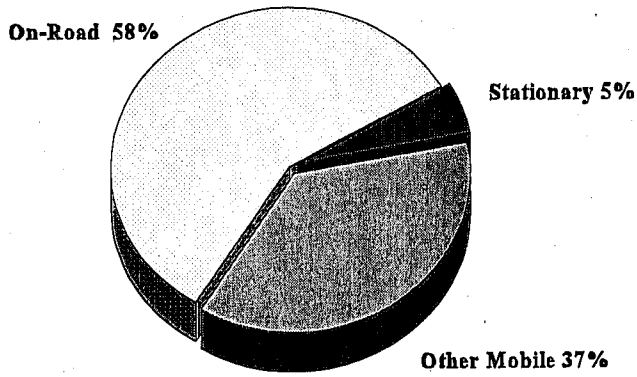
acetaldehyde	hexane
acrolein	inorganic lead
aniline	manganese compounds
antimony compounds	mercury compounds
arsenic	methanol
benzene	methyl ethyl ketone
beryllium compounds	naphthalene
biphenyl	nickel
bis[2-ethylhexyl]phthalate	4-nitrobiphenyl
1,3-butadiene	phenol
cadmium	phosphorus
chlorine	POM, including PAHs
chlorobenzene	and their derivatives
chromium compounds	propionaldehyde
cobalt compounds	selenium compounds
cresol isomers	styrene
cyanide compounds	toluene
dioxins and dibenzofurans	xylene isomers and mixtures
dibutylphthalate	o-xylenes
ethyl benzene	m-xylenes
formaldehyde	p-xylenes

What are the Emissions from Diesel-Fueled Engines?

Diesel-fueled engine exhausts are emitted from three major source categories: mobile sources (on-road vehicles and other mobile sources), stationary area sources (i.e., oil and gas production facilities, shipyards, repair yards), and stationary point sources (i.e., chemical manufacturing, electric utilities). As mentioned above, diesel exhaust is a complex mixture of gases and fine particles. Although we have included emissions of other diesel exhaust pollutants, NO_x, SO_x, reactive organic gases (ROG), CO, and diesel exhaust $PM_{2.5}$ (particulate matter equal to or less than 2.5 microns in diameter), our main focus of the report is on diesel exhaust PM_{10}.

Based on a 1995 emissions inventory, we estimate that approximately 27,000 tons of diesel exhaust PM_{10} (particulate matter equal to or less than 10 microns in diameter) are emitted into California's air each year. On-road mobile sources (heavy-duty trucks, buses, light-duty cars and trucks) contribute the majority, or approximately 15,680 tons per year (tpy) (58 percent) of total diesel exhaust PM_{10} emissions in California. Other mobile sources (mobile equipment, ships, trains, and boats) contribute about 9,820 tpy (37 percent), and stationary sources contribute the remaining 1,400 tpy (5 percent). The figure below shows the percentage of emissions that each major source category contributed in 1995. The revised estimates reflect the results from the new motor vehicle emissions inventory model EMFAC7G1.0.

Sources of Diesel Exhaust PM_{10} Emissions for 1995



Since a majority of the diesel exhaust particles emitted from diesel engines are smaller than 2.5 microns in diameter (or about 94 percent), the emissions inventory estimate is similar to the diesel exhaust PM_{10} estimate. Based on a 1995 emissions inventory, we estimate that approximately 26,000 tpy of diesel exhaust $PM_{2.5}$ are emitted into California's air each year. This is about 3 percent less than the estimated diesel exhaust PM_{10} inventory. The ARB staff also estimates that emissions from diesel exhaust contribute about 3 and 8 percent of the total PM_{10} and $PM_{2.5}$ inventories, respectively.

We have also estimated 1995 emissions from other diesel exhaust pollutants: NO_x, SO_x, ROG, and CO. Based on the 1995 emissions inventory, we estimate that approximately 415,000 tons per year (tpy) of diesel exhaust NO_x, 28,000 tpy of SO_x, 41,000 tpy of ROG, and 188,000 tpy of CO are emitted into California's air each year.

Are Emissions of Pollutants in Diesel Exhaust Expected to Change in California?

Yes. Pollutant emissions from diesel engines have been reduced substantially over the past 20 years. The pollutants in diesel emissions have, and will continue to, decline overall because of improvements in engine design and emission control technology, and the use of reformulated diesel fuels. Consequently, future exposures to particulate matter in diesel exhaust will be lower than current exposures. However, the magnitude of these reductions in exposure will be gradual because of the long life of existing heavy-duty diesel engines and because emission reductions will be somewhat offset by continued growth in vehicle use. The Board has adopted several emission standards and regulations to reduce diesel exhaust particulate emissions, NO_x, and SO_x from many

types of diesel-fueled vehicles and utility engines. These standards and regulations reduce criteria pollutants in diesel exhaust and include:

- a requirement for low sulfur/low aromatic diesel fuel that reduces particulate matter, NO_x, and SO_x emissions from many mobile sources (October 1993);
- emission standards that restrict the amount of particulate matter emitted by new diesel-fueled cars, trucks, urban buses, and heavy-duty trucks (phased-in from 1982 through 1996);
- emission standards for NO_x emissions by diesel vehicles, heavy-duty trucks, and urban buses (phased in from 1984 through 1998);
- the roadside testing of heavy-duty on-road vehicles for excessive particulate matter emissions (1991) and a requirement for fleet inspection and maintenance of heavy-duty vehicles (anticipated to begin in summer 1998); and
- emission standards that restrict the amount of particulate matter and NO_x that can be emitted from many 1995 and newer diesel-fueled utility engines.

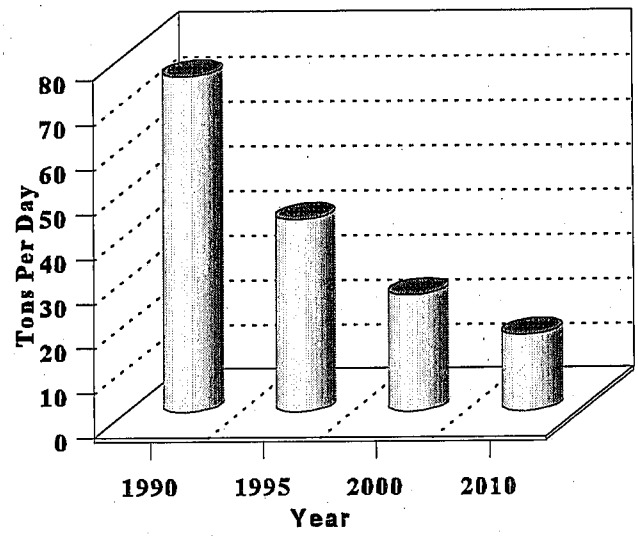
As stated above, significant progress has been made as a result of the above emissions standards and fuel regulations. For example, since 1988, particulate matter emission standards from new heavy-duty diesel truck engines have been reduced by over 85 percent, and urban bus engines by over 90 percent. Emissions of NO_x from heavy-duty diesel truck emissions have also been reduced since the mid-1980's by over 20 percent, and with the implementation of the new 2004 federal standard (discussed below) the NO_x concentrations will have been reduced by over 60 percent from levels in the early 1980's. SO_x emissions have also decreased due to the reduction of sulfur content in fuels by over 80 percent in 1993. Although much work has been done, the ARB staff will need to continue to evaluate the need for further controls to ensure that the expected increase in the number of vehicles on the road and vehicle miles traveled (VMT) does not cancel out the progress that has been made.

Although these regulations were adopted to provide reductions in criteria pollutant emissions, reductions in toxic pollutants, primarily those associated with diesel particulate matter, will also occur.

Emissions of on-road mobile source diesel exhaust PM_{10} in California are expected to decline by approximately 80 percent from 1990 until about 2010 as a result of mobile source standards and regulations already adopted by the ARB (see figure below). The expected reduction is mainly due to adopted diesel vehicle emission regulations, even though both the number and VMT of heavy-duty trucks and off-road diesels are expected to increase during this period. We estimate the number of heavy-duty trucks and the VMT by these trucks in California to increase by about 70 percent and 60 percent, respectively, from 1990 to 2010. Similarly, NO_x

emissions from on-road diesel vehicles have been reduced since 1990, and will continue to be reduced through 2010 because of new NO_x engine emission standards (discussed below).

Diesel Exhaust PM_{10} On-Road Vehicle Emissions Projections



In 1995, as part of California's need to reduce ozone precursors in order to attain national ambient air quality standards, the U.S. EPA, the ARB, and the leading manufacturers of heavy-duty engines signed an agreement to reduce engine emissions of NO_x and hydrocarbons. In this "Statement of Principles," the signatories agreed to pursue new heavy-duty diesel engine standards that will cut oxides of nitrogen emissions from new trucks and buses in half beginning in 2004. This will further decrease emissions of NO_x and particulate matter from the formation of nitrates from atmospheric reactions of NO_x from heavy-duty diesel engines.

In addition, and in order to address the growing concern over the adverse health effects of fine particles, the U.S. EPA has promulgated a new National Ambient Air Quality Standard for $PM_{2.5}$. On July 18, 1997, the annual $PM_{2.5}$ federal standard became 15 $\mu g/m^3$ with a 24-hour $PM_{2.5}$ federal standard of 65 $\mu g/m^3$. How these new fine particle standards will impact future controls of diesel exhaust particulate matter emissions is not known. However, because of their size, diesel exhaust particulate matter is a larger portion of the $PM_{2.5}$ inventory (as compared to PM_{10} inventory), which focuses primarily on the fine particles emitted from combustion sources. As a comparison, ARB estimates that emissions from diesel exhaust particulate matter contribute about 3 percent and 8 percent of the total PM_{10} and $PM_{2.5}$ inventories, respectively.

Is Research Being Done to Investigate the Effects of Pre-October 1993 Diesel Fuel Versus Post-October 1993 Reformulated Diesel Fuels?

Yes. To investigate effects of recent California regulations on diesel fuel content, the ARB has funded several contracts to speciate toxic components in diesel exhaust. During a pilot study in 1995 (Phase 1), researchers from the University of California (UC) Davis collected and developed methods to analyze vapor-phase and particle-phase samples from the tailpipe of a heavy-duty diesel bus using conventional (pre-October 1993) and reformulated diesel fuels. Phase 2 of the study, conducted by researchers at the College of Engineering, Center for Environmental Research and Technology (CE-CERT), UC Riverside, and UC Davis, applied and extended the methods developed in Phase 1 to characterize the differences and toxic components of diesel exhaust, both particle and gas phases, from a heavy-duty diesel engine operating on a pre-October 1993 diesel fuel, on post-October 1993 fuel that meets the 10 percent low aromatic specification, and on an alternative low aromatic complying diesel fuel. Testing was conducted from December 1996 to January 1997 and results became available in the spring of 1998. The CE-CERT report was approved by the ARB's Research Screening Committee on April 3, 1998.

The CE-CERT study was planned as a scoping study. It should be emphasized that the study design did not allow the resulting data to be used to obtain statistically robust conclusions (due to use of only one engine and the limited number of data points for each target analyte/fuel combination). In particular, additional data would need to be collected from other types of engines and driving conditions. Rather, the data collected was intended to be used to characterize the influence of diesel fuel formulation on the emissions of toxic species and to assist in the design of more comprehensive studies.

The results from this study show comparable criteria pollutant reductions to what was estimated in the ARB staff report for the 1988 diesel fuel regulation. The low aromatic and alternative formulation fuels exhaust showed reductions in the particulate matter (PM) and NO_x emission rates as required by the ARB regulation.

A comparison of the milligram to milligram per cubic meter emission profiles using the three different fuels showed the presence of the same toxic substances, and a similar distribution of toxic substances, but with a few substances showing much different emission rates. The low aromatic and alternative fuels resulted in lower emission rates for some particle- and vapor-phase PAHs including anthracene, benz[a]anthracene, dibenzo[a,h]pyrene, napthalene, and biphenyl. In addition, higher mutagenic activity was observed in both the particle- and vapor-phase collected from pre-October 1993 fuel than from the low aromatic and alternative formulation fuels. However, the differences are not statistically significant.

The CE-CERT study also tested for dioxins, with the purpose to identify and attempt to quantify dioxins for method development. The results show that dioxins are present in both the old and the new fuel. However. the results are qualitative only and indicate the need for further method development.

How do We Estimate Exposure to Diesel Exhaust?

Exposures to diesel exhaust are difficult to precisely quantify because of its complex composition, and because many of its components are also emitted from other sources, such as tobacco smoke, manufacturing emissions, and woodsmoke or formed through atmospheric photochemical processes. No single constituent of diesel exhaust serves as a unique marker of exposure, although fine particles and elemental carbon have been used as surrogates of exposure to diesel exhaust PM. Consequently, many researchers have used the particles in diesel exhaust to quantify exposure to whole diesel exhaust. This is the method employed by ARB staff.

As described above, diesel exhaust particles are primarily composed of aggregates of spherical carbon particles coated with organic and inorganic substances. Many of these substances are mutagenic, cytotoxic, or carcinogenic. Moreover, evidence has indicated that the particles themselves may have intrinsic toxic and carcinogenic properties, although the exposure actually experienced in most relevant health studies, both human and animal, has been to whole diesel exhaust. As indicated above, we have used the particle fraction of the exhaust as the basis for estimating the public's exposure to the toxic substances in diesel exhaust. However, until more research is done to determine the cause of the toxicity in diesel exhaust, the identification of whole diesel exhaust is appropriate.

In addition, since the exposure estimates in the report are based on primary emissions of diesel exhaust particles alone, the estimates are conservative and underestimate the public's actual exposure to diesel exhaust as a whole. The estimates in this report do not account for pollutants emitted from diesel engines that may form particulate matter in the atmosphere or other routes of exposure to diesel exhaust such as deposition on water and vegetation.

What Is the Estimated Outdoor Population-Weighted Average Air Concentration of Diesel Exhaust in California?

Staff of the ARB used receptor modeling techniques, which include chemical mass balance results from several studies where chemical speciation of ambient data was performed, ambient PM_{10} monitoring network data, and the 1990 PM_{10} emissions inventory, to estimate statewide outdoor population-weighted air concentrations of diesel exhaust PM_{10}. The ARB used the 1990 PM_{10} inventory for the basis for calculating the statewide exposure to diesel exhaust PM_{10} because it would best represent the emissions sources in the years when the ambient data were collected for the chemical mass balance studies. The estimated population-weighted average outdoor diesel exhaust PM_{10} concentration in California in 1990 is 3.0 micrograms per cubic meter ($\mu g/m^3$). (Stratified by air basin, the estimated outdoor population-weighted concentrations of diesel exhaust PM_{10} range from 0.2 $\mu g/m^3$ in the Great Basin Valley to 3.6 $\mu g/m^3$ in the South Coast Air Basin.)

We have also estimated outdoor exposure concentrations for 1995 based on linear extrapolations from the 1990 to the 1995 emissions inventory. In doing this calculation, it is assumed that the changes in a specific source category, due to growth or emission control, will not greatly alter the distribution of emissions from that source category, and thus affect the outdoor concentration estimate. The estimated 1995 outdoor population-weighted concentration

in California is 2.2 $\mu g/m^3$. Several independent studies have reported outdoor diesel exhaust PM_{10} concentrations consistent with our estimates (see Chapter V, Table V-1).

What are the Projected Outdoor Population-Weighted Average Diesel Exhaust PM_{10} Air Concentrations in California?

The ARB staff has projected population-weighted average outdoor diesel exhaust PM_{10} concentrations for the years 2000 and 2010. The projected outdoor concentrations from primary sources with respect to the 1990 base year emissions inventory were made using linear modeling techniques. The projected outdoor concentrations are based on the projected total emissions inventory of diesel exhaust PM_{10} emissions including on-road mobile, off-road mobile, and stationary sources. Based on emissions inventory projections, staff estimates that population-weighted outdoor diesel exhaust PM_{10} concentrations are 1.8 $\mu g/m^3$ for 2000 and 1.7 $\mu g/m^3$ in 2010. This represents a 43 percent reduction, from 3.0 $\mu g/m^3$ in 1990 to 1.7 $\mu g/m^3$ in 2010. The reductions from 1990 levels are due primarily to ARB- and U.S. EPA-adopted emissions standards and fuel reformulations.

Are There Estimates of Indoor Air Exposure to Diesel Exhaust Particulate Matter?

Yes. To estimate Californians' exposures to diesel exhaust particles, ARB staff used estimates of population-weighted outdoor diesel exhaust particle concentrations for 1990 in a model that can estimate indoor air concentrations, population indoor air exposure, and total air exposure. The model, called the California Population Indoor Exposure Model (CPIEM), was recently developed under contract to ARB to improve estimates of population exposures to toxic air pollutants (see Chapter V, section F). The model uses relevant data (such as distributions of California building air exchange rates, adult and children's activity patterns data, and population-weighted air concentrations of diesel exhaust particles) as inputs to develop indoor concentration estimates and population exposure estimates across all environments.

The average indoor diesel exhaust particle concentrations estimated by the model ranged from 1.6 ($\pm$ 0.7) $\mu g/m^3$ in office buildings to 3.0 ($\pm$ 1.1) $\mu g/m^3$ in industrial plants and inside vehicles. These estimates were combined with activity pattern data in the model to estimate Californians' exposures across all enclosed environments. The exposure modeling results indicate that Californians were exposed to average diesel exhaust particle concentrations of 2.0 ($\pm$ 0.7) $\mu g/m^3$ in enclosed environments in 1990.

We have also estimated average 1995 indoor exposure concentrations as a result of comments we received on the May 1997 draft report. These estimates were not developed using the CPIEM model as were the earlier estimates for the 1990 baseline year. Instead, to provide parallel indoor exposure estimates for 1995, the ratio of the 1990 average indoor exposure concentration estimate (2.0 $\mu g/m^3$) to the 1990 population-weighted average outdoor concentration (3.0 $\mu g/m^3$) was calculated (2.0/3.0) and applied to the estimated 1995 population-

weighted average outdoor concentration of 2.2 $\mu g/m^3$. Thus, the 1995 estimated average indoor exposure concentration is two-thirds of 2.2 $\mu g/m^3$, or approximately 1.5 $\mu g/m^3$.

What is the Population's Estimated Total Air Exposure Concentration of Diesel Exhaust Particulate Matter in California?

The CPIEM was also used by ARB staff to estimate the population's total air exposure to diesel exhaust particles by combining indoor and outdoor air exposure estimates. The model uses the distributions of indoor concentrations, outdoor concentrations, and data on Californians' activity patterns to develop time-weighted population exposure estimates. The population time-weighted average total diesel exhaust particulate air exposure concentration across all environments (including outdoors) is estimated to be 2.1 ($\pm$ 0.8) $\mu g/m^3$ in 1990. We have also estimated total air exposure for 1995 using the same method as used above to calculate the 1995 average indoor exposure concentration estimate. The ratio of the 1990 average total air exposure concentration and the 1990 outdoor population-weighted average estimate is 2.1/3.0. Thus, the 1995 estimated average total air exposure concentration is 2.1/3.0 of 2.2 $\mu g/m^3$ (1995 population-weighted outdoor estimate), or approximately 1.5 $\mu g/m^3$.

These total exposure estimates are believed to underestimate Californians' actual total exposures because insufficient data are available on levels of diesel exhaust particles along roadways, railroad tracks, and inside vehicles to allow such near-source, elevated exposures to be estimated for the population.

What are the Projected Indoor and Total Diesel Exhaust PM_{10} Exposure Concentrations in California?

The staff used the same method for estimating future average indoor and total air exposure concentrations for 2000 and 2010 as was used above in calculating the 1995 indoor and total air exposure levels. We used the same ratio for 1990 indoor to outdoor exposure levels of 2.0/3.0 or two-thirds, and multiplied this by the projected population-weighted outdoor diesel exhaust PM_{10} estimates of 1.8 $\mu g/m^3$ for 2000 and 1.7 $\mu g/m^3$ for 2010. The results estimate average indoor exposure concentrations to be about 1.2 $\mu g/m^3$ for 2000 and 1.1 $\mu g/m^3$ for 2010.

To project the average total air exposure concentrations for 2000 and 2010, we used the same ratio (2.1/3.0) used above to estimate the 1995 average total air exposure concentration. We multiplied 2.1/3.0 by the projected population-weighted average outdoor diesel exhaust PM_{10} estimates of 1.8 $\mu g/m^3$ for 2000 and 1.7 $\mu g/m^3$ for 2010. The results estimate average total air exposure concentrations to be about 1.3 $\mu g/m^3$ for 2000 and 1.2 $\mu g/m^3$ for 2010.

The indoor air and total air exposure concentrations developed for the years 2000 and 2010 are current best estimates but include an amount of uncertainty that cannot be quantified readily. However, the 2000 and 2010 projections most likely underestimate future population exposure to diesel exhaust particles. This is so because those projections are based on the 1990 modeling

estimates, which are believed to underestimate exposures because no data for high exposure environments were available for use as inputs to the models.

What Are the Near-Source Exposures to Diesel Exhaust in California?

Near-source exposures to diesel exhaust occur near busy roads and intersections where diesel vehicles are operating. We also expect higher than average concentrations of diesel exhaust near oil and gas production areas, railroad yards, shipping docks, and other stationary point and area sources where diesel engine use is common. These near-source exposures to diesel exhaust could result in an increased potential health risk for exposed individuals.

In December of 1993, the ARB conducted a study to determine the PM_{10} concentrations due to the primary emissions of diesel exhaust particles near a freeway. Results indicate that diesel exhaust PM_{10} concentrations range from background ambient levels to up to 10 $\mu g/m^3$ for a 24-hour period. This is about five times above the 1995 outdoor ambient air concentration of 2.2 $\mu g/m^3$ and about six times the 1995 total air exposure estimate of 1.5 $\mu g/m^3$. (see Chapter V, section E).

Are There Other Routes of Exposure to Diesel Exhaust?

Yes. Diesel exhaust emissions can deposit onto water, soil, and vegetation. Californians can be exposed to diesel exhaust concentrations through these other media and through airborne exposures to re-entrained dust. However, because substance and site-specific data for these other media are unknown, we could not estimate multi-pathway exposure to diesel exhaust. More research is needed in this area.

What is the Persistence of Diesel Exhaust in the Atmosphere?

As noted above, diesel exhaust is a complex mixture of substances, and each substance will remain in the air or react with other substances according to the substance's individual chemical properties. Diesel particles are typically smaller than 1 micron and are expected to remain in the air for about 10 days, provided that the weather is dry. Diesel particles are effectively removed by precipitation and persist for much shorter periods during these events.

Diesel exhaust also contains organic components which often coat the surface of particles or are found in the gas. Once in the air, they can react with sunlight or with other pollutants to form new substances whose chemical compositions determine their persistence in the atmosphere. One such example would be the gas phase PAH. Because we have used the particulate phase compounds of diesel exhaust to estimate exposures for this risk assessment, we have not documented the persistence of these individual organic substances in the atmosphere in this report.

What are the Health Effects of Exposure to Diesel Exhaust?

The health effects information has been reviewed and evaluated as described below. The information is divided into non-cancer health effects from short-term exposure and long-term exposure, reproductive and developmental effects, immunological effects, genotoxic effects, and cancer health effects.

What is Known about the Non-Cancer Health Effects from Short-term Exposure to Diesel Exhaust?

Human

In experimental studies, healthy subjects have shown increased symptoms of irritation and compromised pulmonary function after short-term exposure. Additional studies have shown that diesel exhaust particles influence localized immunological components involved with allergic reactions. There have also been cases of newly developed asthma reported in workers exposed to diesel exhaust.

The available data from studies of humans exposed to diesel exhaust are not sufficient for deriving an acute non-cancer health risk guidance value. While the lung is a major target organ for diesel exhaust, studies of the gross respiratory effects of diesel exhaust in exposed workers have not provided sufficient exposure information to establish a short-term non-cancer health risk guidance value for respiratory effects.

Animal

The inhalation or direct application of diesel into the respiratory tract of animals in acute and subchronic studies induced inflammatory airway changes, lung function changes, and increased susceptibility of exposed animals to lung infection. The morphological effects observed in the lungs of animals in chronic inhalation exposures are mainly related to chronic inflammatory responses. These changes include thickening of the alveolar epithelium, increase in lung weight, infiltration of macrophages, fibroblasts and proteins into the alveolar septa, and glandular metaplasia.

What is Known about the Reproductive and Developmental Effects from Exposure to Diesel Exhaust?

Studies on induced heritable point mutations and sperm abnormalities following diesel exhaust exposure were negative in rats, mice, and monkeys, though sperm anomalies were noted in exposed hamsters. Data of the effects of diesel exhaust exposure on female reproductive capacity are limited but potential effects on corpora lutea and mating period have been suggested in laboratory rodents.

No teratogenic effects of diesel exhaust exposure were shown in rabbits. Delayed ossification of the thoracic region has been noted in rats following exposure to very high exposure levels. Exposure to diesel exhaust during the neonatal developmental period of rodents induces

neurobehavioral and neurophysiological effects, but does not affect general lung development. Other organ systems have not been evaluated.

Generational studies conducted in rodents revealed that inhalation exposure to diesel exhaust causes increases in lung weight in all generations examined. Evaluation of other parameters produced inconclusive results.

The available literature does not provide sufficient information to determine whether or not diesel exhaust exposure induces reproductive, developmental or teratogenic effects in humans.

What is Known about the Non-Cancer Health Effects from Long-term Exposure to Diesel Exhaust?

Human

Occupational studies showed that the removal of diesel exhaust particles from workplace air improved the pulmonary function of workers. In miners, long-term studies have provided limited evidence of greater incidence of cough and phlegm among those exposed to diesel exhaust than among those not exposed. Most of the epidemiologic studies did not find an excess of chronic respiratory disease associated with diesel exhaust. These studies all had limitations, such as small number of subjects, limited exposure information, and insensitive measures, which clearly reduced their ability to detect adverse effects. The available data from studies of humans exposed to diesel exhaust are not sufficient for deriving a long-term non-cancer health risk guidance value.

Animal

Animal data indicate that chronic respiratory disease can result from long-term exposure to diesel exhaust. In rats, laboratory studies have shown that exposure to diesel exhaust can decrease resistance to infection and increase chronic inflammation. Rats, mice, rabbits, guinea pigs, and other primates all exhibit significant adverse pulmonary noncarcinogenic effects from long-term exposures to diesel exhaust.

Based on the animal studies, the U.S. EPA determined a chronic inhalation Reference Concentration value of 5 micrograms per cubic meter for noncancer effects of diesel exhaust. A U.S. EPA Reference Concentration or California Reference Exposure Level (REL) of a chemical is an estimate, with uncertainty spanning perhaps an order of magnitude, of the air concentration below which no noncancer adverse health effects are likely to occur from lifetime exposure. This estimate takes into consideration persons who may be more sensitive than others to the effects of the chemical. The report provides a range of estimated threshold levels for non-cancer effects which support the recommendation of 5 micrograms per cubic meter as the California REL.

What is Known about the Immunological Effects of Diesel Exhaust?

There are a number of review articles which postulate that air pollution, particularly diesel exhaust particles, plays a role in the increasing prevalence of asthma and other allergic respiratory

disease. The discovery of the role of diesel particulates and their polycyclic aromatic hydrocarbon fraction in augmentation of allergic responses to specific antigens in humans and animals is relatively new.

Diesel exhaust exposure can result in measurable increases in immunoglobulin E and immunoglobulin G antibody production, perturbed immunological cytokine regulation, localized inflammation and eosinophilic infiltration in lung and respiratory tract tissues, particularly when the exposure accompanies other known respiratory allergens. In human subjects and in human cells, diesel exhaust particulate stimulated immunoglobulin E antibody production and increased messenger RNA for the pro-inflammatory cytokines. Co-exposure to diesel exhaust particulate and ragweed pollen was reported to significantly enhance the immunoglobulin E antibody response relative to ragweed pollen alone. Diesel exhaust particulate also enhanced the immunoglobulin E antibody and cytokine production response to ovalbumin and Japanese cedar pollen in animal models and increased nasal hyperresponsiveness to histamines. There is some evidence that production of reactive oxygen species may be involved in the asthma-like symptoms produced in mice by diesel exhaust particulate exposure. Although none of the studies evaluated have been designed to yield quantitative estimates of diesel particle concentrations for the purposes of determining a reference exposure level, the potential relevance of these immunological endpoints to public health is very high, due to reports of large numbers of individuals with respiratory allergies and asthma in urban areas.

What is known about the Accumulation of Diesel Exhaust in the Lung?

Clearance of deposited particles is conveniently characterized in three phases. Early clearance appears to be similar across the species studied. Intermediate-phase clearance rates appear to vary depending on species, dose and duration of exposure. Lung clearance rates for the mouse and rat are approximately four times greater than those for humans and dogs. The late phase occurs during impaired removal. Repeated exposure to sufficiently high concentrations results in excessive particulate loading. Particle-laden macrophages exhibit decreased mobility and phagocytic activity resulting in sluggish transport of particles from the lungs. Experiments in the rat have shown that at high exposures the mass of diesel exhaust particles remaining in the lung does not appear to reach a steady state as exposure time increases. This overload phenomenon occurs because lung clearance does not keep up with deposition. Normally, clearance rates in the human lung are substantially slower than clearance rates in rat lungs. This information was considered in interpreting the animal and human studies and in extrapolating the results to ambient concentrations. Information on toxicokinetics is used to predict a relationship between exposure and accumulation of diesel soot in the lung.

What do the Genotoxic Health Effects tell us about the Cancer Causing Potential of Diesel Exhaust?

Much of the information regarding genotoxicity has been obtained using diesel exhaust particles or extracts of diesel exhaust particles. Diesel exhaust particles or their extracts are mutagenic in bacteria (*Salmonella typhimurium* and *E. coli*) and in several mammalian cell systems (Chinese hamster ovary, V79, BALB/c3T3, L5718Y mouse lymphoma, human

lymphoblasts). Diesel exhaust particles or their extracts induce chromosome aberrations, aneuploidy, and sister chromatid exchange in rodent and human cells in culture. Diesel exhaust particles and their extracts are also capable of inducing cell transformation. Diesel exhaust particles or their extracts can also produce superoxide and peroxide radicals and inhibit the antioxidant enzymes responsible for radical scavenging. Diesel exhaust particles have also been shown to cause an increase in 8-hydroxydeoxyguanosine (8-OHdG) adducts in calf thymus DNA *in vitro* and in lung DNA from mice exposed *in vivo* by intratracheal instillation. Both diesel exhaust particle extracts and the semivolatile phase of diesel exhaust have dioxin receptor (*Ah* receptor) binding affinity. Exposure to diesel exhaust particulate matter can cause unscheduled DNA synthesis *in vitro* in mammalian cells. DNA adducts have been isolated from calf thymus DNA in vitro and mouse lung DNA following intratracheal instillation.

Some information regarding genotoxicity also has been obtained directly from diesel exhaust exposures. Whole diesel exhaust has been demonstrated to induce gene mutations in two strains of *Salmonella*. Inhalation exposure to diesel exhaust results in DNA adduct formation in rodents and monkeys. Increased levels of human peripheral blood cell DNA adducts are associated with occupational exposure to diesel exhaust. The genotoxic effects of diesel exhaust may be involved in the initiation of pulmonary carcinogenesis in humans.

Diesel exhaust clearly contains genotoxic substances. Furthermore, diesel exhaust particles and diesel exhaust extracts have been established to be genotoxic. The bioavailability of these genotoxins has been questioned. Several lines of evidence suggest bioavailability. First, the *in vitro* genotoxic activity of diesel exhaust particulates dispersed in pulmonary surfactant exhibited similar activity to extracts of diesel exhaust particles. Second, inhalation exposure of rats and monkeys to diesel exhaust results in DNA adduct formation and *in vitro* exposure of rat tissues to diesel exhaust induces unscheduled DNA synthesis. Third, DNA adducts have been associated with occupational exposure to diesel exhaust. Fourth, urinary metabolites of PAHs have been found following exposure of rats to diesel exhaust. Preliminary evidence indicates the same may be true for humans. Consequently, it appears that organic chemicals adsorbed onto the particles, particularly the genotoxic components, are likely to be bioavailable in humans.

What Does the Composition of Diesel Exhaust tell us about the Cancer Causing Potential?

Many carcinogenic compounds are found in diesel exhaust. Compounds found in the vapor phase include benzene, formaldehyde, 1-3-butadiene, and ethylene dibromide. At least 16 hydrocarbons that are classified as possibly carcinogenic (IARC Classification 2B) to humans are adsorbed on the exhaust particles. Additionally, benzo[a]pyrene, benz[a]anthracene, and dibenz[a,h]anthracene, which are classified as probably carcinogenic to humans (IARC Classification 2A), are adsorbed on the particles. A report of work at the U.S. EPA concluded that adding the carcinogenic effect estimated for all these compounds does not account for all of the carcinogenic effect of the whole diesel exhaust. While the vapor includes numerous cancer causing compounds, several animal studies found tumors only from exposure to the diesel exhaust particles. Three rat bioassays found that filtration of the exhaust, in order to remove particles, eliminated any significant tumor response to exposure. In contrast, a study in one strain of mice found similar increased incidences of malignant tumors in filtered and unfiltered exhaust groups. However, a similar study in the mouse by the same investigators did not find such an effect.

A general issue with regard to characterizing the toxicity of diesel exhaust is the variability of exhaust composition among types of engines and over different driving (or other use) conditions. However, findings suggest that variability in toxicity may be small when the health evaluation is based on the concentration of particulate matter.

What are the Cancer Health Effects from Exposure to Diesel Exhaust?

Human

Over 30 epidemiological studies have investigated the potential carcinogenicity of diesel exhaust. The epidemiological evidence primarily concerns cancer of the lung. The question whether diesel exhaust causes lung cancer has been addressed by both industry-based cohort studies and case-control studies as well as population-based studies of lung cancer. There were no published industrial hygiene measurements of diesel exhaust exposures for any of the study populations. Therefore, exposures have generally been defined indirectly by occupation and duration of employment.

Animal

Results of inhalation bioassays in the rat have demonstrated the carcinogenicity of diesel exhaust in test animals. All seven studies in rats using exposure concentrations of greater than or equal to 2.2 mg/m^3 of whole diesel exhaust (time-weighted average equivalent to approximately 1 mg/m^3), and using observation periods of approximately 2 years or longer, reported statistically significant excesses of lung tumors. Four studies in rats at lesser exposures or with shorter observation periods were inconclusive or negative. The finding that the rat lung also develops cancers in response to other particulates, carbon black and titanium dioxide, indicates that diesel exhaust particulate matter may be important to the carcinogenicity of diesel exhaust in the rat. Studies in mice have mixed results. Unfiltered diesel exhaust significantly increased lung tumor

incidence in female Strong A mice, female Sencar mice, and female NMRI mice. In female Strong A mice, however, the highest exposure resulted in a decrease in lung tumor incidence relative to controls. Exposure of female NMRI mice to filtered diesel exhaust has produced both positive and negative results. Other studies in mice are negative. All three studies in hamsters were negative. Negative results in hamsters are consistent with the finding that, unlike rats, hamsters do not demonstrate increases in DNA adduct formation following a 12-week exposure to diesel exhaust particulate matter (see Section 5.4 of Part B).

The mechanisms by which diesel exhaust induces lung tumors in rats are not certain. Several hypotheses have been proposed. One hypothesis invokes the genotoxicity of the compounds condensed on the surfaces of the diesel exhaust particle. This hypothesis suggests the operation of a general mechanism shared with humans and the absence of a dose-response threshold. Another hypothesis is that diesel exhaust leads to oxidative damage to DNA by a mechanism other than particle-induced inflammation. A third hypothesis is that the particulate nature of diesel exhaust is responsible for its carcinogenicity. The inflammatory response to any fine particle could, at high lung burdens, cause cellular proliferation and thereby increase the chance of DNA replication before repair. This last mechanism could possibly operate in humans but may suggest a dose-response threshold. More than one mechanism may be involved as there is some evidence for each hypothesis.

Other Agencies and scientific bodies?

The National Institute of Occupational Health and Safety (NIOSH) first recommended that whole diesel exhaust be regarded as a potential occupational carcinogen based upon animal and human evidence in 1988. It is our understanding that they are undertaking additional analyses and may undertake additional epidemiological studies. The International Agency for Research on Cancer concluded that there is sufficient evidence for the carcinogenicity of whole diesel engine exhaust in experimental animals and that there is limited evidence for the carcinogenicity of whole diesel exhaust in humans. On that basis, IARC concluded that diesel engine exhaust is probably carcinogenic to humans and classified diesel exhaust in Group 2A. Based upon the IARC findings, in 1990, the State of California under the Safe Drinking Water and Toxic Enforcement Act of 1986 (Proposition 65) identified diesel exhaust as a chemical known to the State to cause cancer. The 1998 draft U.S. EPA document similarly concluded that diesel exhaust be considered a "probable" human carcinogen by inhalation exposure and best fit into cancer weight-of-evidence category B1 according to EPA's 1986 Cancer Risk Assessment Guidelines. This conclusion evolves from positive yet "limited" evidence in the human studies, a "sufficient" level of evidence in bioassays, and consideration of the supporting information from mutagenicity and genotoxicity data.

The Health Effects Institute (HEI) and the World Health Organization (WHO) also evaluated the carcinogenicity of diesel exhaust (HEI, 1995; WHO, 1996). The HEI and WHO both found that the epidemiological data are consistent in showing weak associations between exposure to diesel exhaust and lung cancer. The HEI and others have also considered that the absence of reliable exposure data, inability to control for some confounders, and questions about one's ability to estimate a dose-response relationship in the epidemiological studies limits the ability to use them for quantitative risk assessment. The HEI found that the carcinogenicity of diesel exhaust

had been convincingly demonstrated in rats. However, the HEI also concluded that the rat lung response was likely related to an overload of the rat clearance mechanism. HEI found that there was evidence indicating the effect was specific to the rat and therefore cautioned against extrapolating the rat findings to humans exposed at ambient levels.

What does the Meta-Analysis Show?

In order to summarize quantitatively the overall and occupation-specific risks from the epidemiological studies, a meta-analysis was conducted (see Appendix C of Part B). This meta-analysis provides strong support for the hypothesis that occupational exposure to diesel exhaust is associated with an increased risk of lung cancer. Pooled relative risk estimates from 30 studies clearly reflect the existence of a positive relationship between diesel exhaust and lung cancer in a variety of diesel-exposed occupations. Another independently conducted meta-analysis was published recently, also reporting a persistent positive relationship between occupational diesel exhaust exposure and lung cancer that could not be attributed to potential confounding by cigarette smoking (see Section 6.2.2 of Part B).

Based upon the epidemiological review and meta-analysis, these epidemiological studies provide evidence consistent with a causal relationship between occupational diesel exhaust exposure and lung cancer. The majority of these studies have reported elevated estimates of relative risk for lung cancer, many of which are statistically significant. These studies, on average, found that long-term occupational exposures to diesel exhaust were associated with a 40 percent increase in the relative risk of lung cancer. This level of increase is generally characterized as a weak association, which theoretically may diminish the evidence for causality. Weak associations require greater scrutiny because they can be greatly affected by other influencing factors (that is, confounders and biases). The OEHHA analysis considered the potential influence of other factors as discussed below.

OEHHA analyzed the lung cancer findings from the various studies for consistency and found that the association was unlikely to be due to chance. The association held up when the various studies were analyzed in a variety of ways (for example, by occupational subgroup, by type of study design, by control for smoking). Several studies provide evidence of exposure-response relationships. This association is supported when adjusting for the most important potential confounder, cigarette smoking. OEHHA also analyzed the data for publication bias - that is, whether small studies providing null or inconclusive results were systematically less likely to be published than other studies. Though some publication bias cannot be ruled out, OEHHA staff found that the results could not be explained by publication bias.

Is there Evidence of Other Cancers from Exposure to Diesel Exhaust?

There is some evidence linking occupational exposure to diesel exhaust with increased risk of bladder cancer. However, the evidence linking diesel exposure and bladder cancer is not as extensive or compelling as that for lung cancer.

What is the Potential Cancer Risk from Exposure to Diesel Exhaust?

Sources of other information for cancer risk estimates

In general, risk assessments can use carcinogenicity data from animal or human studies. While data exist for the carcinogenicity of diesel exhaust in the rat, the uncertainty in the application of the rat findings to humans is substantial. The scaling of such important characteristics as clearance rates, the presence or absence of a threshold for onset of carcinogenic effects, or the possible presence of multiple carcinogenic mechanisms all contribute to the uncertainty. The present lack of knowledge about how the carbon core of the diesel exhaust particle contributes to carcinogenicity also adds to the uncertainty about the scaling from rats to humans. For example, it has been suggested that rodent lung tumor induction by particles such as carbon black and titanium dioxide may not be relevant to human cancer risk if due to an overwhelming of particle clearance and no other tumor responses are noted (Commission on Risk Assessment and Risk Management, 1996).

There are data from occupationally exposed populations useful for quantitative risk assessment. The principal uncertainties in using the human data are the representativeness of the study populations for the general population, the choice of the analytical models, and the actual exposure that resulted in the increased incidence of cancer.

On balance, the human data lend more confidence in the prediction of human risks than the data from the rat studies because of the uncertainties of extrapolating from rats to humans, especially in the context of the substantial particle effect. Thus, OEHHA preferred to derive the human risk estimates based only upon the epidemiological findings and not the rat data.

Estimates using epidemiological studies

The Garshick *et al.* (1987a) case-control study and the Garshick *et al.* (1988) cohort study of U.S. railroad workers were used to estimate the risk of lung cancer in the general population due to diesel exhaust. These two studies were selected for quantitative risk assessment because of their quality, their apparent finding of a relationship of cancer rate to duration of exposure and because of the availability of measurements of diesel exhaust among similar railroad workers from the early 1980's in other studies. The case-control study (1987) has an advantage in providing direct information on smoking rates, while the cohort study (1988) has an advantage of smaller confidence intervals in the risk estimates.

Range of risk estimates based on epidemiological studies

Calculations using the two studies of Garshick *et al.* (1987a, 1988) and the reanalyses of the individual data of the Garshick *et al.* (1988) cohort study in Appendix D provide a number of estimates of unit risk. The relative risks reported in these studies were related to estimates of the actual exposures to estimate potential cancer risks. Because of uncertainties in the actual workplace exposures, OEHHA developed a variety of exposure scenarios to bracket the possible exposures of interest. The following Table 1-1 presents the range of resulting estimates of cancer risk.

Table 1-1. Summary of Cancer Unit Risks According to Study, Exposure Assumptions, and Modeling Approaches.

	95% UCL Cancer Unit Risk $(\mu g/m^3)^{-1}$	Upper Limit of Cancer Risk per Million per Microgram of Diesel Exhaust Particulate in a Cubic Meter of Air Exposure Over a 70-year Lifetime
Garshick *et al.* (1987a) Case Control[1]		
Scenario[2]		
A	2.4×10^{-3}	2400
B	1.8×10^{-3}	1800
C	1.0×10^{-3}	1000
D	6.6×10^{-4}	660
E	3.6×10^{-4}	360
Garshick *et al.* (1988) Cohort Study (Chapter 7)[3]		
Scenario		
A	1.8×10^{-3}	1800
B	1.4×10^{-3}	1400
C	8.2×10^{-4}	820
D	5.1×10^{-4}	510
E	2.8×10^{-4}	280
Garshick *et al.* (1988) Cohort Study (Appendix D)[4]		
Scenario A		
general multiplicative model	1.9×10^{-3}	1900
biologically based [5]	3.8×10^{-4}	380
Scenario C		
general multiplicative model	7.2×10^{-4}	720
biologically based [5]	1.3×10^{-4}	130

[1] ***Using published slope coefficient for hazard on years to diesel exhaust as described in Section 7.3.3 of Part B.***

[2] ***A*** *Ramp pattern of exposure plateauing in 1959 at the 1980 exposure level of 50* $\mu g/m^3$

B *Roof pattern of exposure peaking in 1959 at twice the 1980 exposure level of 40* $\mu g/m^3$

C *Roof pattern of exposure peaking in 1959 at 3-fold the 1980 exposure level of 50* $\mu g/m^3$

D *Roof pattern of exposure peaking in 1959 at 3-fold the 1980 exposure level of 80* $\mu g/m^3$

E *Roof pattern of exposure peaking in 1959 at 10-fold the 1980 exposure level of 50* $\mu g/m^3$

[3] ***Using individual data to obtain a slope for hazard on years of exposure to diesel exhaust as described in Section 7.3.4 of Part B.***

[4] ***Applying time varying concentrations to individual data to obtain a slope of hazard on exposure as described in Appendix D., of Part B.***

[5] ***6th/7 stage model.***

[6] ***7th/7 stage model.***

biologically based [6]	1.5×10^{-4}	150

Risk estimates from other agencies or scientific bodies

OEHHA's unit cancer risk estimates are not directly comparable to those developed by the WHO and the U.S. EPA. WHO selected a range of risks based only upon animal studies and then estimated risks based on human studies. U.S. EPA most recently proposed a range of risks including both human and animal studies. OEHHA selected a range of risks based only on human studies, but risks based upon animal studies were also calculated. The respective numerical values of risks for animals were quite similar across the three agencies. Risks based on human studies were also quite similar for the three agencies, approximately 10-fold higher than risks based upon animal studies.

What are the Uncertainties in Calculating a Potential Cancer Risk?

Results based on the human data and those based on the animal data are both subject to considerable uncertainty. The principal uncertainties in using the rat data are their application to humans in terms of response, the choice of dose-response model to extrapolate the risk to environmental concentrations, the presence or absence of a threshold for response, and the range of dose extrapolation involved. This is why OEHHA has chose to use only the unit risk estimates based on human data in the final range of unit risks.

The principal uncertainties in using the human data are the representativeness of railroad workers for the general population, the choice of the analytical model, and the lack of knowledge of the exposure history of the railroad workers including possible exposure to unknown confounders. The historical reconstruction here is based upon the Woskie *et al.* (1988b) exposure data for railway workers and the rate of dieselization for U.S. railroads. Using a range of reduced emission assumptions, alternate exposure patterns are considered. This reconstruction takes into account to some degree the likely higher exposure levels in the past. If actual exposures were higher than assumed here, then our estimates of the risk would be lower. And if exposures were lower, then the estimated risks would be higher. The range of extrapolation from these estimated occupational exposure levels to the California total air exposure of 1.54 μg diesel exhaust particulate/m^3 is not large.

A number of individuals and organizations have indicated that the epidemiological studies are limited in their application to environmental risk assessment. OEHHA recognizes that the limited exposure information available does contribute to the overall uncertainty of the dose response risk assessment for diesel exhaust based upon the epidemiological findings. However, the overall magnitude of the associated uncertainty is not unduly large. The greater than usual uncertainty in the exposure estimates is substantially offset by the much smaller than usual range of extrapolation from the occupational exposures of interest to the ambient levels of concern here. The availability of human data obviates the need to use animal data, thus avoiding uncertainties of

animal-to-human extrapolation. OEHHA provided a tabular range of risk so as to fairly capture the scope of the uncertainty in these analyses.

HEI is proceeding on a Diesel Epidemiology Project to develop new information to improve risk assessments of diesel exhaust. It is possible the results of the project may improve our ability to quantitatively assess the potential public health risk of exposure to diesel exhaust.

Has a Threshold Level for Diesel Exhaust Exposure Been Identified?

Based upon on information available, the report could not identify a threshold below which no significant adverse health effects are anticipated. It has been suggested that information based on the rat data suggested the presence of a threshold. However, the same data suggests that the rat data may not be relevant to humans.

Why Does the Staff Recommend that the Board Identify Diesel Exhaust as a TAC?

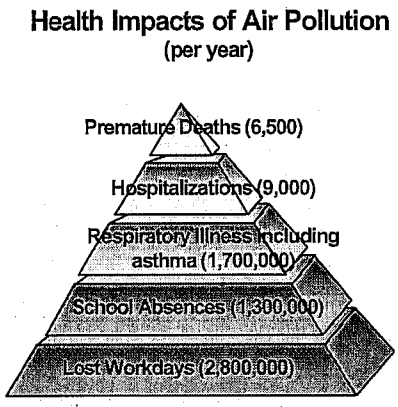
Based on the information available on diesel exhaust-induced noncancer and cancer health effects and the results of the risk assessment, diesel exhaust meets the definition of a TAC. We believe that at current ambient concentrations, diesel exhaust may cause an increase in the likelihood of cancer. Therefore, we conclude that diesel exhaust meets the legal definition of a TAC which is an air pollutant "which may cause or contribute to an increase in mortality and serious illness, or which may pose a present or potential hazard to human health" (Health and Safety Code section 39655).

California Environmental Protection Agency
AIR RESOURCES BOARD

AMERICAN LUNG ASSOCIATION of California

Recent Research Findings:

Health Effects of Particulate Matter and Ozone Air Pollution, January 2004



Although air pollution levels in California have improved significantly in the past few decades due to aggressive controls on vehicles and industry, many Californians still breathe the worst air in the nation. California's climate and geography are conducive to the formation and accumulation of air pollution (especially in Los Angeles and the Central Valley). These factors, combined with increasing population and economic growth, the dramatically increasing number of vehicle miles traveled, and other factors, make it difficult to reduce pollution levels. Higher and longer summer temperatures have also worsened smog problems. The concentrations of several pollutants not only exceed California's health-based standards, but are often measured at levels up to two or three times the standards.

Premature deaths linked to particulate matter or "PM" are now at levels comparable to deaths from traffic accidents and second-hand smoke (CARB 2002a). One of the most dangerous pollutants, fine particulate matter (e.g., from diesel exhaust and fireplace soot) not only bypasses the body's defense mechanisms and becomes embedded in the deepest recesses of the lung, but also can disrupt cellular processes. Population-based studies in hundreds of cities in the U.S. and around the world have demonstrated a strong link between elevated particulate levels and premature deaths, hospital admissions, emergency room visits, and asthma attacks. Groundbreaking long-term studies of children's health conducted in California have demonstrated that particle pollution may significantly reduce lung function growth in children (Peters et al. 1999, Avol et al. 2001, Gauderman et al. 2002).

Another dangerous pollutant is ozone. Ozone is a powerful oxidant that can damage the respiratory tract, causing inflammation and irritation, and induces symptoms such as coughing, chest tightness, shortness of breath, and worsening of asthma symptoms. Ozone in sufficient doses increases the permeability of lung cells, rendering them more susceptible to toxins and microorganisms. The greatest risk is to those who are more active outdoors during smoggy periods, such as children, athletes, and outdoor workers. Exposure to levels of ozone above the current ambient air quality standard leads to lung inflammation and lung tissue damage, and a reduction in the amount of air inhaled into the lungs. Recent evidence has, for the first time, linked the onset of asthma to exposure to elevated ozone levels in exercising children (McConnell 2002). These levels of ozone also reduce crop and timber yields, damage native plants, and damage materials such as rubber, paints, fabric, and plastics.

Scientific research is constantly uncovering new information on air pollution health effects and the mechanisms by which pollutants damage the heart and lungs and contribute to asthma attacks and premature death.

Air Pollution Causes Premature Death

Attaining the California PM standards would annually prevent about 6,500 premature deaths, or 3% of all deaths. These premature deaths shorten lives by an average of 14 years. This is roughly equivalent to the same number of deaths (4,200 – 7,400) linked to second-hand smoke in the year 2000. In comparison, motor vehicle crashes caused 3,200 deaths and homicides were responsible for 2,000 deaths (CARB 2002a, and CDHS 2000).

Air Pollution Leads to Hospitalizations and Emergency Room Visits

Attaining the California PM and ozone standards would annually prevent approximately (CARB 2003a):

- 4,000 hospital admissions for respiratory disease.
- 3,000 hospital admissions for cardiovascular disease.
- 2,000 asthma-related emergency room visits.

Air Pollution Contributes to Respiratory Illnesses and Cancer

Attaining the California PM and ozone standards would annually prevent about (CARB 2003a):

- 400,000 cases of lower respiratory symptoms (such as a cough) in children ages 7-14.

- 400,000 cases of upper respiratory symptoms (such as, runny nose, wet cough, and burning, itching, red eyes) in children ages 9-11.
- 8,000 cases of chronic bronchitis.
- 500,000 cases of respiratory illnesses (including colds and flus) in adults ages 18-65.
- 350,000 asthma attacks (all ages).

Although statistics are not available for cases of lung cancer caused by all air pollutants, it is estimated that exposure to diesel PM causes about 250 excess cancer cases per year in California (CARB 2000). A recent study provides evidence that exposure to particulate air pollution is associated with lung cancer (Pope et al. 2002). This study found that residents who live in an area that is severely impacted by particulate air pollution are at risk of lung cancer at a rate comparable to non-smokers exposed to second-hand smoke. Definitive lung cancer mortality numbers as a result of air pollution cannot yet be determined, but this study found an approximately 16 percent excess risk of dying from lung cancer due to fine particulate air pollution.

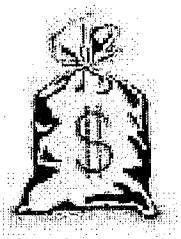
Air Pollution Contributes to Cardiac Illnesses

The hearts of sensitive individuals (for example, the elderly) may be affected when they breathe in fine particulate matter. One study shows that individuals with existing cardiac disease can be in a potentially life-threatening situation when exposed to high-levels of ultrafine air pollution (Peters et al. 2001). Fine particles can penetrate the lungs and may cause the heart to beat irregularly or can cause inflammation, which could lead to a heart attack. Understanding this link is extremely important in quantifying the detrimental health effects of air pollution.

Air Pollution Contributes to School Absences

On a statewide basis, 1.3 million school absence days would be avoided annually if the current levels of ozone were reduced to attain the established 1-hour state standard (CARB 2004).

Air Pollution is Costly



Air pollution can and does have a serious impact on the State's economy. Figures related to asthma costs and the valuation of air pollution exposure are significant and staggering. Analyses indicate that the benefits of California's air quality program exceeds the costs by a ratio of about 3 to 1 (CARB 2003c).

In 1998, it was estimated that asthma costs in California totaled $1.3 billion with hospitalizations and medications representing the largest direct expenditure (Asthma and Allergy Foundation of America 1998). Adult asthma patients spent an average of $5,000 annually on medical expenses, lost wages, transportation, asthma-control products, and other asthma related expenses (Cisteinas et al. 2003).

Furthermore, an annual value of over $3.5 billion is associated with hospitalizations and the treatment of major and minor illnesses, and about 2.8 million lost workdays each year, are all related to air pollution exposure in California. In addition, the value of premature deaths resulting from exposure to air pollution in excess of the State's PM2.5 standard is $43 billion (CARB 2003a, CARB 2003b, CARB 2002a, U.S. EPA. 1999).

Sensitive Groups Advised to Restrict Activities

Sensitive groups, including the elderly, people with heart or lung disease, children and infants, can be at increased risk of experiencing harmful effects from exposure to air pollution. Sensitive individuals are advised to restrict certain activities when pollution levels are elevated. Recently, the number of unhealthy days in some areas of California (based on California standards that are more health-protective than federal standards. (CARB 2003b) has been approximately one out of every three days for ozone (CARB 2002b).

Unhealthy Days in 2002		
	South Coast Air Basin	San Joaquin Valley
Days Above National 8-Hour Ozone Standard	96	125
Days Above State 1-Hour Ozone Standard	116	127

People in almost every area in California are exposed to PM levels over the current standards.

State's Population Living in Areas that Exceed PM2.5 Air Quality Standards*	
Annual National PM2.5 Standard	61%
Annual State PM2.5 Standard	89%

*Based on the proposed designations for PM2.5.

Summary of the Health Effects of Air Pollution

Particulate Matter Health Effects	Ground-level Ozone Health Effects
➢ Aggravated asthma ➢ Increased respiratory symptoms ➢ Chronic bronchitis ➢ Increased respiratory and cardiovascular hospitalizations ➢ Decreased lung function in children ➢ Lung cancer ➢ Premature deaths	➢ Aggravated asthma and possibly new cases of asthma ➢ Reduced lung capacity ➢ Increased susceptibility to respiratory illnesses ➢ Increased respiratory and cardiovascular hospitalizations

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